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STRUCTURES AND FUNCTIONS OF SELECTIVE ATTENTION

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Structures and Functions of Selective Attention*,1

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Abstract

A principle problem of neuropsychology is to relate the neural structures damaged in traumatic brain injury with their functions in the cognitive tasks of daily life. This lecture reviews evidence that elementary operations of cognition as defined by cognitive studies are the level at which the brain localizes its computations. Orienting of visual attention is used as a model task. The component facilitations and inhibitions in visual orienting are related to neural systems through the study of focal neurological lesions.

Visual orienting is a part of a more general selective attention system that also involves orienting to language. Our ability to be aware of and to act upon target events depends upon the connections of posterior orienting systems to anterior systems involved in target detection. We have examined these pathways in studies of focal changes in cerebral blood flow during performance of language tasks. Although we do not have a general analysis of the mental operations performed by these anterior systems, there is some evidence relating the dorsolateral prefrontal and areas of the medial surface to aspects of focal selection.

One way to study the generality of the attentional system developed in this lecture is to examine putative deficits of attention in disorders such as schizophrenia, depression and closed head injury where the organic basis for the deficit is largely unknown. Our preliminary studies of schizophrenia are used to support the utility of the joint functional and structural analysis

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Annotation

Annotation

^{*} This is a draft of a lecture to be given as the Master Lecture in the Neuropsychology of Attention at the American Psychological Association meeting, New York, August, 1987. To appear in a volume on Neuropsychology to be published by APA.

The central problem of neuropsychology is to understand the relationship between everyday life performance and the neurosystems that support it. On the one hand, clinical neuropsychologists are faced with neuroimages that provide a picture of the locations of lesions and on the other hand, they must discuss with patients, relatives and insurance companies, likely deficits in performance that will be seen in daily life.

Among the many deficits found in brain injury is the ability to maintain performance in the face of competing information. This requires selection of information among competing events. Selective attention is an old topic within experimental psychology (James, 1890; Titchener, 1908) and most frequently refers to performance when there are conflicts between signals. Attention has the role of selecting some signals for higher levels of processing, including conscious processing, while preventing access of other signals to those same high levels of processing. Selective attention plays an important role in most cognitive tasks including pattern recognition, reading and mental imagery (see Posner, 1982 for a historical review).

During the last dozen years it has been possible to work out some aspects of the neural structures involved in selective attention based upon work with humans (Posner, Walker, Friedrich and Rafal, 1984) and alert monkeys (Mountcastle, 1978; Wurtz, Goldberg & Robinson, 1980). The research has been accomplished by many different investigators, but studies have used similar tasks and have been constrained by our increased understanding of the anatomy of the visual system (Cowey, 1985) and to some extent of the frontal lobes (Goldman-Rakic, in press). Thus, something of a common overall view has begun to emerge despite remaining conflicts and uncertainties. Edited volumes have summarized this work from anatomical, physiological, neuropsychological and cognitive perspectives (see Posner & Marin, 1985; Berluchi & Rizzolatti, 1987).

The work on spatial attention may serve as a useful model for understanding the way in which cognition is represented within the nervous system. It already has provided a basis for understanding some functions of selective attention such as visual pattern recognition (Treisman, 1987; Prinzmetal, Presti & Posner, 1986) including the recognition of visual words (Posner & Presti, 1987). Research on spatial attention provides a basis for understanding deficits of attention found in such diverse disorders as schizophrenia, depression and closed head injury.

This paper first traces a general framework for connecting cognitive and neural systems of selective attention. Next it reviews effects of unilateral brain lesions on the cognitive operations of visual spatial orienting. Studies discussed in this section show how damage to this system affects pattern recognition. Next evidence relating attention to language and attention to visual locations is reviewed in order to construct a general picture of the structure of the attention system. Finally, our knowledge about the structure and function of attention is applied to a condition whose organic basis is unknown - schizophrenia.

I. Framework

It is useful to view the connection between cognitive systems and neurosystems in terms of a very general framework (Posner, 1986). This framework involves five levels of analysis shown in Fig. 1. At the highest

level are tasks of daily life. Cognitive scientists have developed a number of computational models for tasks such as visual imagery (Kosslyn, 1980), reading (Rumelhart & McClelland, 1981) and typewriting (Rumelhart & Norman, 1982). These tasks provide a view of the computations necessary for any electromechanical system to perform the cognitive tasks described. Some of these computational models consist of subroutines that operate on symbolic representation, labeled here as "elementary operations". They resemble the types of operations studied by cognitive experiments of the last twenty years (Posner, 1978). Each operation can be specified in terms of the input to the operation and its output. Sample operations include match, store, zoom, compare, engage and move. These operations sometime serve as labels on the box models of information flow that dominate textbooks of cognitive psychology.

FIG. 1

In recent years a new form of computational model has arisen in several areas of psychology (see McClelland & Rumelhart, 1986 for a review). parallel distributed or connectionist models do not discuss performance in terms of elementary operations directly but instead refer to facilitations and inhibitions between levels. Fortunately, as we have learned to measure elementary operations in chronometric experiments, we find that they can be specified in terms of component facilitations and inhibitions in performance. My own work has often attempted to describe mental operations in terms of these time-locked facilitations and inhibitions (Posner, 1978; Posner & Snyder, 1975) in reaction time. Methods for making such measurements have been described and have been widely applied (Jonides & Mack, 1984; Neely, 1986; Posner, 1978; Taylor, 1977). A great deal is known about how such measures can be taken and what pitfalls there are in using them (Jonides & Mack, 1984). The use of the words "facilitation" and "inhibition" in connectionist models and in the description of the components of elementary mental operations are biased to make one inquire as to whether such patterns are related to the activity of populations of nerve cells that might perform the computation. To what extent do our findings on facilitation and inhibition in the performance domain and in connectionist models relate to changes in populations of nerve cells? This relationship is a central question for the neuropsychology of cognition. If it is possible to move from the level of facilitation and inhibition in performance to the level of neurosystems, one can then see how it is possible to go from an understanding of lesions in an area of the nervous system to predictions about normal cognition.

The study of visual spatial attention has been extremely important to this enterprise. Visual spatial attention can be studied in people and in alert monkeys. The presence of an animal model provides opportunity to determine whether results obtained from performance studies of normal humans converge with those using single cell methodology. In so far as this link can be established, it is possible to move from the general study of neurosystems, as can be done in human beings by studies described here, to the study of individual nerve cells.

II. Facilitation and Inhibition in Visual Spatial Attention

In attempting to work out a complex system like selective attention, it is important to study experimental situations or "model tasks" that define what we

mean by the phenomenon and allow us to study it in simple forms. An important aspect of selective attention is orienting to a source of visual signals. In this area, similar model tasks have been used in studies of animals, normal humans and patients (Posner & Marin, 1985; Berluchi & Rizzolatti, 1987). The importance of studying covert shifts of attention is the hope that the mechanisms involved in these shifts of attention will help us understand more general problems of selectivity in other modalities and in memory.

A very simple model task is illustrated in Fig. 2. The subject is cued to shift attention covertly to a visual location eccentric of where the eyes are currently fixed. In these experiments the cue may be an event that occurs at the location to which the subject is to attend (brightening of a box) or it may be a symbolic instruction (e.g. arrow at fixation) that informs the subject where to shift attention.

Fig. 2

Many experiments have now been performed with both of these forms of cueing. The results for tasks illustrated in Fig. 2, uniformly show an advantage for the cued location over the uncued location that is closely time-locked to the occurrence of the cue. This relative facilitation has been measured in reaction time (Posner, 1980), probability of correct detections for near threshold stimuli (Bashinski & Bachrach, 1980), and increased electrical activity at the cued in comparison with the uncued location (Mangun, Hansen & Hillyard, 1986).

Two interrelated issues in interpretation of these findings remain in dispute. First, is the relative facilitation of the cued location a genuine improvement of information coming from the cued location or a reduction or inhibition of information coming from all other locations? Second, is the extent of the facilitated area. Most experiments seem to be consistent with the idea that facilitation occurs not only at the attended location but also in a gradient over a range of adjacent locations (Downing & Pinker, 1985, Rizzolatti, Riggio, Dascola, & Umilta, 1987). The size of the area facilitated depends in part on the degree of eccentricity from the fovea and in part upon the complexity of the information in the visual field. How large a part of the visual field is represented by the focus of attention? This has been a widely disputed issue. For example, Hughes & Zimba (1985), have argued that attention acts simply by inhibiting the hemifield to which one is not attending. have found a facilitation localized to the neighborhood of the target and increasing with eccentricity from the fovea, with an inhibition stronger once one has crossed the midline (Downing & Pinker, 1985). These disputes indicate the complexity of the overlapping processes that accompany a shift of attention.

My basic approach to these complexities has been to develop a functional model that can both account for these findings and conforms to other properties associated with attention. According to this functional viewpoint (Posner & Cohen, 1984), three basic components are involved when attention is summoned by a cue located in the neighborhood of a likely target. These components combine to determine the net increase in efficiency at the cued location. First, the cue increases alertness because a target is now expected. It is known from previous work that alertness is not spatially selective (Posner, 1978) and

works to potentiate all targets following the cue. Second, the cue initiates a spatially selective movement of visual attention to the cued location. Such attention shifts are not fully automatic in sense of being unavoidable (Posner, Cohen, Choate, Hockey & Maylor, 1984), but they occur with little effort if the subject does nothing to avoid them (Jonides, 1981).

Third, the occurrence of a cue in the periphery initiates two forms of inhibition. The first, called "cost", is a consequence of orienting attention to the cue. Once attention is engaged at the cued location, all other locations will be handled less efficiently (inhibited) than if no such orienting had occurred because one must first disengage from the cued location before moving to targets at other locations. This form of inhibition is spatially selective only in the sense that it is not present within the focus of attention. A second form of inhibition also occurs. This is called "the inhibition of return" (Posner & Cohen, 1984). The inhibition of return depends upon the act of orienting to a spatial location (Maylor, 1985), but it is most clearly shown if one summons attention to a location and then returns it to a neutral location. The efficiency of the previously cued location is reduced with respect to comparable locations in the visual field for several seconds. The overlap between facilitation due to orienting of attention and the specific inhibition of a cued location helps to explain conflicts in the literature. Sometimes a cued location is handled more efficiently than other locations, sometimes less efficiently, depending upon the balance between the facilitation due to orienting of attention and the inhibition due to the reduction of efficiency of returning attention to an already cued location.

Are there any ecological advantages to this very complex constellation of internal events by a cue? Our theory rests on our finding that the relative facilitation obtained from a peripheral cue moves with the eyes as though it mapped in retinal coordinates (Posner & Cohen, 1984). This effect is not on the retina since it can be obtained in stereoscope. However, it preserves the coordinates of the retina as do many visual images at cortical levels. Inhibition of return on the other hand, does not move when the eyes do, it behaves as though it were dependent on the coordinates of the environment. When we move our eyes, the objects of the world appear to maintain their locations, thus many psychological phenomena maintain the coordinates of the environment as we move about it. It seemed to us of basic importance that one of our effects (facilitation) is retinotopic and the other, (inhibition of return) is environmental in this sense.

According to our view the facilitation effect serves to give priority to targets during a visual fixation. It allows us to give momentary priority to an object in the visual field as, for example, when we carefully examine the nose within a face. If the task demands high acuity, we are likely to move our eyes to the examined location and thus, produce a reorienting of attention back to the fovea. In reading, for example, the reduction of acuity with eccentricity may be the cause of the eye movement (Morrison, 1984). Attention allows a temporary emphasis outside of the fovea, and it is crucial as a guide to the occulomotor system to tell us where to move the eyes next.

We speculate that inhibition of return evolved to maximize sampling of novel areas within the visual fields. Once the eyes move away from target location, events occurring at that environmental location are inhibited and one is less likely to move the eyes back to them (Posner, Choate, Rafal & Vaughn, 1985). This reduces the effectiveness of an area of space in summoning

attention and serves as a bias for favoring fresh areas in which no previous targets have been presented. The long lasting nature of inhibition of return insures that two to three eye movements are biased against a return. The organization of facilitation and inhibition outlined above seems to represent an exquisite functional adaptation to the needs of the visual world.

We review the operations involved in our model task in Fig. 3. The top of the figure indicates the occurrence of a visual cue. The bottom indicates a set of partially sequential but overlapping mental operations induced by the cue. According to this diagram,

Fig. 3

the cue produces a non-spatially specific alerting effect which serves to interrupt ongoing performance. The cue also leads to calculation of its coordinates and in turn produces a disengagement of attention, a movement to the location of the cue and subsequent engagement of the target. If the subject's attention is withdrawn from the cue to another location, we can measure the inhibition at the target location that we call inhibition of return. Single cell recordings in monkeys and the study of patients with restricted neurological lesions can be used to examine the neurosystems that support each of these operations. The basic argument developed in the next section is that widely separated neurosystems are involved in the computation of these various mental operations.

III. Deficits of Orienting from Focal Lesions

Several areas of the monkey brain have cells whose firing rates are enhanced selectively when the monkey's attention is directed to targets in their receptive fields (Wurtz, Goldberg & Robinson, 1980). In one of these areas, the superior colliculus, the selective enhancement occurs only when attention is directed overtly via eye movement. In a second area, the posterior parietal lobe, selective enhancement occurs when attention is directed overtly or when the monkey is required to maintain fixation while attending covertly to a peripheral stimulus. The third area of selective enhancement lies between the midbrain and cortical projections in the thalamic nuclei known as the pulvinar (Petersen, Robinson & Morris, 1987). enhancement appears to be restricted to these three areas. The single cell results allow us to ask about the relationship between modulation of cellular activity and patterns of facilitation and inhibition found in our work with humans. In this sense they provide an opportunity to connect the last row of Figure 1 (cellular level) with the facilitatory and inhibitory performance changes described for visual spatial orienting. These connections are fundamental to our effort to see if the nervous system localizes the components of cognitive operations.

There is a long clinical history documenting the finding that lesions of the posterior part of one hemisphere can cause a severe deficit in reporting information on the side of space opposite the lesion (DeRenzi, 1982). Neglect of visual information contralateral to the lesion occurs most strongly when patients are confronted with simultaneous lateralized visual stimuli and stimuli contralateral to the lesion are frequently not reported (extinguished). The phenomena of neglect can arise from unilateral lesions of the midbrain and

thalamus as well as from a variety of cortical lesions. However, clinical observations seem to suggest parietal lesions on the right side as the most frequent area of damage leading to neglect and extinction (DeRenzi, 1982).

In recent years a number of these parietal patients have been studied in experiments using cues such as those described in the previous section (Baynes, Holtzman & Volpe, 1986; Morrow & Ratcliff, 1987; Nagel-Leiby, Buchtel & Welch, 1987; Posner, et al, 1982; Posner, et al, 1984; Posner, et al, 1987). The studies have been uniform in showing a particular type of deficit present in patients with right parietal lesions. These patients have a general advantage in reaction time for those targets that occur ipsilateral to the lesion in comparison to those that occur contralateral to the lesion. However, for many parietal patients there is little or no difference between the two types of targets if they follow a cue at the same location (Valid Trial). When attention is drawn to either side, these patients have nearly equal ability to detect the target at the cued location. Thus the ability to engage the target once attention is properly directed is not necessarily interrupted by parietal lesions although it is affected in many patients.

Striking results occur on trials when attention is cued to the side of the lesion and the target is presented to the side opposite the lesion. In some cases, targets show extinction, that is, targets are missed entirely by the subject (Posner, Cohen & Rafal, 1982). In other cases, targets are not completely excluded from consciousness, but show greatly delayed reaction time, sometimes two or three times the normal reaction time. The results suggest that this elevation in latency is simply a less severe form of complete exclusion from consciousness. Patients who miss signals completely when they remain present in the field only briefly will report them when they remain present but with greatly increased latency. The idea that a latency increase is a less severe form of difficulty than extinction fits with the account of covert orienting in normals discussed previously.

The pattern of increased reaction time to contralateral targets following miscues does not depend upon the miscue being ipsilateral to the lesion. Indeed, the increases in reaction time occur in both visual fields when the subject has to produce a covert movement in a contralesional direction from the cue to the target (LaDavas, 1987; Posner, et al, 1987). For patients with right parietal lesions, leftward movements from cue to target are longer than rightward movements to the same target. These findings suggest that the main deficit in parietal patients occurs in the disengage operation. It may be instructive to review the logic. On validly cued trials there is only a modest difference in reaction time on the two sides. Moreover, the improvement in reaction time following a valid cue appears to be about the same on the two sides of the field. We argue that this reduction is due to a shift of attention, thus many parietal patients (those with no difference between valid RTs between the two visual fields) are able to shift attention equally well to the two sides. However, once attention is engaged either at fixation or in either visual field, RTs to targets that lie in a contralesional direction are greatly elevated for even those patients with no differences on valid trials. Why should this be? Why should contralesional targets be at so great a disadvantage following a cue at another location? We reason that it must be because the parietal lesion has a special affect on disengaging attention.

This specific deficit in the disengage operation for contralateral targets found in parietal patients has been confirmed in a number of experiments

(Baynes, et al, 1986; Morrow & Ratcliff, 1987). There are several remaining complexities, that have not been successfully resolved. Using a central cue, Nagel-Leiby, et al. (1987) has found differences between males and females and that in some cases, frontal patients show more severe deficits than parietal patients. In addition, Morrow & Ratcliff (1987), who confirmed our basic result with right parietal patients, have found little deficit in left parietal patients and also found a similar pattern to the right parietal patients in one frontal lobe patient. The unique status of the parietal lobe that appeared clear in our earlier work seems somewhat in question. The issue may be partly resolved by the widespread effects that occur immediately following a lesion. For some months following an insult to the nervous system, there may be widespread changes in glucose utilization and blood flow over the entire hemisphere (Deuel & Collins, 1984). It is possible that some of the reports from other areas may have arisen because the patients were tested too early. The deficits we have reported persist even when patients are tested years after the stroke. Morrow & Ratcliff (1987) have traced these recovery effects for some months following lesions.

Another reason for finding these effects in frontal patients may be because the spatial attention system is not an isolated module that operates independently of other levels of control. Thus lesions of the frontal lobe may affect the spatial attention system along with a variety of other systems because it influences command systems necessary to allow for the disengagement process. This possibility will be discussed in more detail on page 000. It is not completely clear whether lesions of the left parietal lobe produce an identical pattern with the same strength as lesions of the right parietal lobe. These comparisons are, of course, always between subjects and thus can involve many sources of error not found in the within subject comparisons on which we have mostly relied.

Forms of Neglect

Clinically neglect occurs after a wide variety of lesions. This may be in part because many of the reports of neglect are from studies of patients who are acutely ill and may have widespread metabolic problems following the initial insult. We have so far found only three groups of patients who show systematic deficits in visual spatial orienting even after relatively long periods of time after the lesions. These correspond to areas that give selective enhancement in single cell studies of alert monkeys. The reaction time patterns in these three forms of "neglect" are shown in Figure 4. It shows the reaction times to valid and invalid trials at short intervals between the cue and the target and divides them according to whether the target occurs in the field which is usually neglected (left panel) or the one which has no evidence of neglect (right panel). In the case of parietal lesions the neglected field involves the area of space contralateral to the lesion.

Fig. 4

A second group of patients have progressive supranuclear palsy, with lesions of the midbrain, including the superior colliculus and surrounding areas. These patients show an unique deficit in eye movements, having great difficulty making voluntary eye movements, particularly in the vertical direction. Impairment develops more slowly for horizontal movements. These

patients often come to the neurologist's attention because they neglect the lower part of the visual field. In the case of these patients we have systematically compared attention movements in the vertical direction with those in the horizontal direction (Posner, et al, 1985). The results for these patients are very striking and completely different for those found for parietal patients. As can be seen from figure 4, these midbrain patients have very long reaction times. The long reaction times may be due to the widespread reticular lesions along with the deficits that we have described. However, in the horizontal direction there is clear evidence of a validity effect. Even at short intervals, valid trials are systematically faster than invalid trials. Thus orienting to horizontal targets appear relatively normal. However, in the vertical direction, the validity effect does not emerge until much later. There is no evidence of a validity effect at the fast probe interval shown in Fig. 4 but usually by half a second a validity effect has emerged.

These data are very different from the parietal patients who show a greater than normal validity effect in the neglected field at the earliest intervals. Since the emergence of a validity effect is due to a shift of attention to the cued side, the findings from the midbrain patients suggest a specific delay in their ability to move attention to the target. Hence, if enough time is given following the cue, the vertical and horizontal directions both show validity effects. It appears that the deficit in the midbrain patients is in their ability to move attention covertly in the direction that has the largest eye movement deficit.

An additional finding with supranuclear palsy patients is that they lose inhibition of return in the vertical direction. Although they can move attention to a vertical cue if given sufficient time, they do not show the reduced tendency to return attention to a previously cued location (Posner, et al, 1985). This loss of inhibition of return was unique to these midbrain patients and is not found in control groups with cortical or other subcortical lesions. It fits quite well with the functional theory that identifies inhibition of return with the tendency to move the eyes to novel locations. Deficits in the move and inhibit operations provide more evidence in favor of the idea that specific neurosystems influence different aspects of the set of computations necessary to induce orienting of visual spatial attention.

A third form of "neglect" has been found following thalamic lesions that may involve the pulvinar (Rafal & Posner, 1987). As can be seen from Figure 4, these patients show another pattern of performance deficit, especially long reaction times for the invalid trials on the side opposite the lesion. This effect is similar to that found in parietal patients, although the deficit on invalid contralesional trials does not appear to last as long following the cue in the thalamic patients. Striking in the thalamic patients is that the increase in RT is also quite large for valid trials on the side contralateral to the lesion.

This constellation of deficits for both valid and invalid trials could be consistent with a purely visual defect. However, careful ophthalmologic testing of these patients, particularly in their six month follow-up, showed no evidence of ophthalmologic deficits. The second explanation would be a specific deficit in their ability to engage attention on the side contralesional to the target. This would suggest that these patients cannot use attention to make processing as efficient as it could be when targets that are contralesional. This supports the idea that thalamic lesions produce a

specific deficit in the engage operation and provide some support for a theory of the special role of thalamic areas in control of the attentional spotlight (Crick, 1984). In Crick's view, the thalamus is the area of the brain most likely to be involved in the search of the complex visual field for targets. A deficit in the engage operation would be consistent with this theoretical view.

Figure 4 summarizes three patterns that we have found present for posterior lesions related to aspects of poor RT performance to targets contralateral to the lesion. These include: parietal lesions and the disengage operation; thalamic lesions and the engage operation and finally, midbrain lesions and the move operation. The results do not show complete separation. For example, parietal patients frequently show engage deficits and thalamic lesions also produce disengage deficits as well. The known anatomy and close physiological connections of these areas would lead to the expectation that the three are in close contact. For covert orienting to occur all these operations must be performed. One assumes that the disengage operation begins the sequence, information is then sent to the midbrain to move attention to an already calculated location and when that is completed, it is possible for the system to work through thalamic sites to engage targets. An important point is that the thalamus (particularly the lateral pulvinar) represents an area allowing contact between parietal systems responsible for spatial attention and systems of the brain known to be responsible for pattern recognition. It is clear that patients with lesions of the parietal lobe do show deficits in pattern recognition process and we turn to evidence of this effect in the next section.

IV. Functions of Spatial Attention in Pattern Recognition

According to recent views of the neurophysiology of vision, there are two major systems extending from the primary visual cortex. The first extends from area V1 (striate cortex) to the inferotemporal cortex and is involved in the recognition of objects. The second extends from area V1 into the parietal lobe and is more responsible for localization of information and as we have discussed above, for visual spatial attention (Mishkin, Ungerleider, & Macko, 1983). It is important to ask whether deficits in visual selective attention influence the pattern recognition process, and if so, in what way?

We have developed two different strategies to evaluate this issue. First, cueing in normals can be used to control orienting of attention, and can then explore the effects of such cues on pattern recognition. Second, patients with deficits in visual spatial attention due to specific lesions can be studied. It is possible to ask both whether the cueing known to be responsible for covert visual orienting influences pattern recognition and also whether the presence of lesions in areas related to visual spatial attention influences pattern recognition. The answer to both questions seems to be yes and provides us with information on the relationship of attention to pattern recognition.

According to one recent theory, visual spatial attention has the role of integrating visual features into conjunctions (Treisman, 1987). Individual features of objects such as color, orientation, or motion are to some extent registered in separate spatial maps in monkey cortex. This registration occurs in parallel across the entire visual field. If an object differs from its background by a single feature, it is possible for a person to respond to the presence of that feature rapidly and efficiently without attending to individual items in the field. If, however, the judgment requires the integration of features into a conjunction, such as looking for a red T in a

field of Ts and other red objects, spatial attention is needed and a more serial search is conducted. If normal subjects are cued to a location eccentric of the fovea, both feature and conjunction search are conducted more efficiently at the cued location. However, the effect on conjunction search is far stronger than for feature search (Prinzmetal, et al, 1986; Treisman, 1987). This suggests that although attention can effect the registration of features, it plays a more important role in the recognition of conjunctions.

Similarly, it is possible to study the effects of lesions of the visual spatial attention system on the visual search process. It is well known clinically that right parietal lesions produce a relative neglect of information on the side of space opposite the lesion. Experiments show that both right and left parietal lesions have clear effects on visual pattern recognition (Friedrich, Walker & Posner, 1985). Subjects were presented with two strings of letters, one above the other. The letter strings were identical half the time and half the time differed by a single letter. This difference could be in the beginning of the string, in the middle or at the end of the The subject's task was to press one key if the strings were identical and another if they were not. Subjects were free to move their eyes, and the letters remained present in the visual field until they responded. Left parietal patients showed extreme difficulty when the discrepant letter was at the end of the letter string. Reaction times were nearly 800 msec longer for differences found at the end than at the beginning. Moreover, the subjects frequently missed differences at the end. On the other hand, right parietal patients were slower and made more errors when the differences were at the beginning. This task is an attention demanding spatial search task and shows quite clearly the pattern recognition deficits in the parietal patients. ability to organize and recognize differences on the side of space opposite the lesion is greatly impaired even when they can take the time to move their eyes and examine the stimulus in detail. These were patients far removed from the stroke and showed little evidence of clinical neglect or extinction. Despite the general recovery, the visual search task showed clear deficits.

An important distinction in the study of pattern recognition is between automatic and attended processes. By exploring automatic processes we can examine the operations for which attention is not needed. One process that has been a candidate for "automatic" is the ability of a visual word presented on the fovea to contact its visual, phonological and semantic representations in memory (LaBerge & Samuels, 1974; Marcel, 1983; Posner, 1978). The advantage of an integrated word, even in comparison to individual letters, has been an important theme in cognitive psychology and in recent connectionist models of visual word processing (McClelland & Rumelhart, 1986). It thus became of considerable interest when Sieroff & Michel (In Press) reported that patients who show profound extinction of individual short words when they are presented simultaneously to the right and left visual field showed no evidence of extinction with tachistoscopic presentation of a single word across the fovea, even when it covered the same visual angle as the word pair. Patients with both right and left parietal lesions showed clear evidence of extinction to simultaneous words but even right parietal patients showed little evidence of extinction to the single foveally presented word.

We compared the perception of single eight letter strings (Sieroff, Pollatsek & Posner, 1987) that either formed words or not. Tests of ten right parietal patients presented at bedside with 3 x 5 cards showed that the

patients missed the first few letters of nonwords, but not of words (see Figure 5). This result was confirmed by tachistoscopic testing of right and left parietal patients who were well past the lesion. The results showed that the recognition of the letters in nonwords appeared to depend upon an intact visual spatial attention system, but for words the lesion did not produce any spatially specific deficit. These results fit with the findings in cognitive psychology that word perception is superior to nonword perception. One reason given for the superiority of words is that recognition of visual words might have top down assistance from a visual lexical dictionary (McClelland & Rumelhart, 1986).

Fig. 5

The cueing method can also be used to bias visual spatial attention in normals (Sieroff & Posner, 1987). Thus it should be possible to confirm our patient results by looking at the processing of normal subjects with attention drawn covertly either to the left or right end of strings of letters. To do this, we first presented a digit for 50 msec below the position in which would follow the first or last letter of a 100 msec exposure of a letter string. The results with the normal subjects were were similar to those found with the patients. For words, biasing of attention to the beginning or end made little difference in the parts of the string correctly reported. For random letter strings, the subjects systematically missed information on the side of the word away from the cue. The more word-like the letter string, the less the effect of the cue on the subject's report.

These experiments show quite clearly that visual spatial attention deficits produced by parietal lesions can have very strong effects on pattern recognition. They also suggest that both formation of conjunctions and reports of stimuli making nonword strings are greatly affected by attention. However, for word strings, there is little or no effect of damage to the spatial attention system nor of shifts of spatial attention in normals. Foveal words appear to have automatic access at least to a visual lexicon.

Recent studies of normals using Positron Emission Tomography (PET) to study regional cerebral blood flow during visual language tasks, have provided additional evidence for the rapid packaging of individual letters into word forms (Petersen, Fox, Posner, Mintun & Raichle, 1986). In these studies subjects in separate blocks (a) watched passively while nouns were presented visually once each second, (b) pronounced the nouns, (c) generated uses (verbs) to the nouns. During 40 seconds of the task, regional blood flow was assayed by use of PET. A subtractive technique allowed examination of the neural systems active when either watching words passively or actively responding to them. The passive visual task activated areas of the prestriate cortex as far anterior as the occipital temporal boundary (see Fig. 6). This activation is very different from that found with auditory words (Fig. 6). When the subject was required to pronounce the words or to generate uses for the words, two parts of the anterior cortex (frontal lobe) were activated. One part was left lateralized and seemed specifically related to language (see Fig. 7,8). The repetition task appears to activate areas near and superior to the classic Broca's area. These areas appear to relate to the generation of the articulatory code of the visually presented word. The generate task activates areas more anterior (close to area 45) on the lateral surface that appear to be related to the semantic operations in achieving the use of the presented word. The areas activated in the use generation task for visual and for auditory words are in close proximity but appear separate (Petersen, Fox, Posner & Raichle, 1987). The second set of anterior areas are on the medial surface and do not necessarily seem to be language related. These areas include the supplementary motor area and the cingulate cortex. We believe these areas may be parts of the anterior focal attention system that is discussed in the next section. Roland (1985) has reported several areas that seem to accompany almost all forms of cognitive activity. The areas to which he refers appear to be somewhat more anterior to the ones we have found active, but differences in our techniques may account for anatomical differences. In any case there do appear to be several candidate areas that may be involved in coordination of attention to visual spatial and language information.

Fig. 6,7,8

The fact that no posterior area other than in the occipital lobe was activated by visual words, whether the subject was passive or whether he was active, suggests that the visual analysis of words must take place within the occipital lobe. This result fits with several findings within the psychological literature. First, it fits well with the results described above in which subjects with right parietal lesions extinguished the left side of nonwords but not of word strings. The lesion result suggests that the word/nonword distinction must be made rather early in the nervous system. Second, models of interactive computations (McClelland & Rumelhart, 1986), require intimate feedback from higher levels to lower ones. The rich feedback available in the occipital lobe would make an ideal basis for this system.

Third, many cognitive studies with letters and words conducted in the late 1960s and early 1970s (see Posner, 1978 for a review) argued that visual codes of letters and words had access to output systems. When subjects were required to indicate whether a letter or word pair were physically identical they could do so independently of the names or semantics of the items shown. Nonetheless, words were responded to faster than nonwords. This would require the ability to route visual input to output mechanisms without having to go through phonetic, or semantic systems. As further support for this idea, we (Sandson & Posner, 1987) asked subjects to make lexical decisions about whether or not a string of letters made a word. They did the lexical decision task either alone or while also shadowing a verbal message. We found that priming of the target by an identical immediately prior string (identity priming) was not reduced by shadowing while all forms of semantic priming were reduced by shadowing. This result supports the idea that physical priming involves visual spatial pathways and their connection to output system (in this case manual), while semantic priming involves systems in which visual and auditory input is intermixed.

Although many previous anatomical theories of visual word reading had relied upon information reaching the angular gyrus or Wernicke's area, (Geschwind, 1965) current cognitive literature discusses the use of a purely visual code as a means of accessing semantic memory (See Carr & Pollatsek, 1985 for a review). These PET studies confirm the idea that visual spatial attention is not needed for pattern recognition of individual words outlined in this section, but also suggest the importance for anterior areas in higher levels of attentional control of language. It is to these higher levels of control that we now turn.

V. Common Systems of Attention to Language and Visual Space

In this chapter we have been using two cognitive systems to examine control by attention. These are a visual spatial attention and one which processes language information. The two systems can be viewed in terms of a hierarchy of attentional control systems as shown in Figure 9. Visual spatial attention can be seen as part of a system involving orienting to sensory information. We know that parietal lesions can impair orienting to tactile and auditory information as well as to visual information. Moreover, impairments in different forms of sensory orienting are independent in the sense that auditory and visual extinction are not correlated among patients with parietal lesions (DeRenzi, Gentilini & Pattacini, 1984; Sieroff & Michel, 1987). Similarly, we found that a cue that draws attention to a spatial location was ineffective when the person did not also know the modality of the target (tactile or visual). These findings suggest separate neural systems within the parietal lobe responsible for attention to visual, tactile or auditory modalities.

Fig. 9

On the other hand, it is possible to compare the relative influence of modality (auditory vs. visual) with the influence of the type of cognitive system (spatial or language) in the control of attention. The two cognitive systems correspond to the two major branches of Figure 9. In a series of studies with normals (Posner & Henik, 1983) and patients (Walker, Friedrich & Posner, 1983) we have used a spatial version of the Stroop effect to study this issue.

In these experiments subjects are instructed to respond either to the visual words "left" or "right", to the location of these words on the screen, to visual symbols (arrows pointing to the left or right), or to auditory words ("left" or "right") that might be presented to the left or right ear. In different experiments manual or vocal responses have been used. In work with normals (Posner & Henik, 1983), we compared irrelevant dimensions using either the same cognitive system but a different modality than the attended event, with those in the same modality but a different cognitive system. When a person is to deal with a visual or auditory word the extent of facilitation or conflict in RT from words in the opposite modality is much greater than from spatial locations in the same modality. For example, the auditory word "right" interferes more with processing the visual word "left" than does the location of the visual word on the screen. Stimuli from the same cognitive system, even when they involve different sensory modalities, interact strongly. This motivates the common nodes for language and for space independent of modality (see Fig. 9).

Reading is one task that clearly involves both language and spatial attention since eye movements and higher level semantic codes are both involved. However, the choice of language and spatial attention was designed to allow for the possibility that above the spatial processing needed for foveating visual words, the control mechanisms for the two systems might be quite separate. So far we have shown that the visual spatial attention system includes the posterior parietal lobe, areas of the thalamus and midbrain. We now ask whether this visual spatial attention system is an independent module

that operates on its own or whether it operates in relation to a more complex attentional system that is also involved in the processing of, for example, auditory language.

One way to examine this task is to ask normal subjects and patients with parietal lesions to perform a language task and at the same time, to respond to cues and targets occurring at varying locations in the visual field (Posner, Inhoff, Friedrich & Cohen, 1987). For patients this required a very simple language task in which they listen to twenty words, one every second, and count the number of times the words contain a particular phoneme. While listening to these words, visual cues appeared at two locations in the field and we measured the speed of pressing a key to targets following those cues. The language task retarded the ability of a cue to draw the subject's attention to a location in visual space. These patients show large validity effects, (advantage of the cued location over the uncued one) by 100 msec when performing the spatial task alone, which under dual task conditions no validity effect was found until 500 msec. The same results can be obtained with normal subjects, however, they require a more complicated task than the very simple phoneme monitoring task used with patients. For example, a similar retardation of the cueing effect can be obtained if the subjects are required to count backwards by three (Posner, et al, 1984).

As expected the dual task increases reaction time to the visual spatial processing task. However, it does more than merely increase reaction time, it also retards the validity effect. This suggests that the ability to orient attention is retarded when the person is engaged in a language task. Language tasks interfere with some of the operations necessary to shift visual spatial attention to a cued location. Thus visual spatial attention is not an independent module but shares operation with a more general attention system also involved in the processing of language.

Can we say more about the interaction between visual attention and language processing? The use of patient populations does allow us to show that the interaction between visual spatial attention and language attention does not involve the parietal visual attention system. This conclusion stems from the finding that the parietal lobe lesion produces a deficit in the disengage operation. Patients would have to show a specific slowing on invalid contralateral targets when processing language, if language used the same parietal system. However, when engaged in the language task, patients show little difference in reaction time between targets that are ipsilateral versus contralateral to the lesion. Apparently the disengage deficit is local only to visual spatial attention and is not a general disengage deficit. The results of the PET scanning data support these findings since we find no common posterior areas that are involved in auditory and visual language processing. Thus if one seeks an area that deals with language processing (both in its visual and auditory form), and in visual spatial attention (see Fig. 8), one must move to anterior parts of the brain. Whatever system is involved in processing visual spatial and language information must lie in the frontal lobes and/or their related subcortical areas.

The frontal lobes are currently a very active area of research within neuropsychology. Good summaries of this work are available (Goldman-Rakic, in press). It is well known that lesions of this area can produce devastating effects on human thought and behavior that in one review has been likened to producing a person whose thought and behavior lacks coherence (Duncan, 1986).

One result of including frontal lobe function in the ability to allocate attention to visual space is to reconcile the existing conflicts in the literature. Even if the basic visual spatial attention system is posterior, as we have argued, its control system may lie within the frontal lobes and affect both language and spatial function. Thus findings that neglect can be obtained from frontal lesions may have to do with the command functions that act to allow the posterior areas to function (See Fig. 3). The common finding in experimental psychology that much of our attentive behavior is closely related to motor performance (Allport, 1980) fits with the idea that attentional systems lie in close proximity to symptoms controlling motor output.

Although I believe that midline systems that we have found activated in our PET scanning experiments (See Fig. 7,8) are likely to be part of the focal attention system of the frontal lobe, we do not yet have definitive studies that have localized the different arc computations that performed within the control structures found in the frontal areas. The relationship of computational models of executive function to the complex anatomy of the frontal lobes still remains in the future, although a beginning of this kind of thinking has arisen, particularly on the role of the dorsolateral prefrontal cortex in inhibiting conflicting responses (Diamond, 1987). One must keep in mind the lessons learned from the posterior attention system that such systems involve widely scattered cortical and subcortical sites. As we seek to understand the anterior attention control systems it is likely that we will discover many anatomically distinct areas to be involved.

VI. Applications to Putative Disorders of Attention

The implication of the framework that has organized this lecture is that deficits in mental function must be described both in terms of the elementary operations impaired and of the neurosystems affected. To develop this theme we have been dependent upon cases in which the damaged anatomical area can be observed by neuroimaging. This is traditional neuropsychology. There are many putative disorders of attention, however, in which the underlying neural damage is unknown. These disorders are said to be attentional in the relatively loose sense that they seem to involve the ability of the person to concentrate, to interact appropriately with the environment, and do not seem to be simply due to sensory, motor or general cognitive damage. Four disorders in this category are depression, schizophrenia, closed head injury and attention deficit disorder. In each of these, the literature indicates a disorder of attention and while there are ideas about the organic basis of the disorder it is still is unknown.

I would like to use schizophrenia as a model illustrating how the framework developed in this chapter may serve to guide research relating cognitive and neural systems. My interest in schizophrenia began with a study using Positron Emission Tomography in never medicated schizophrenics (Early, Reiman, Raichle & Spitznagel, 1987) showing a left basal ganglia abnormality. This anatomical result, together with the widely held belief that schizophrenia was a disorder of attention (Mirsky & Duncan-Johnson, 1986), led us (Posner, Early, Crippin & Reiman, 1987) to examine the operations of visual spatial attention among schizophrenics. The hypothesis was that there would be a right visual field deficit (because of the left hemisphere abnormality found in PET) that would occur under conditions in which attention had first been drawn to the left visual field (because of the attentional nature of the disorder). We used our

standard visual cueing method. Our initial results have confirmed this hypothesis (See Fig 10).

Fig. 10

The advantage of using this simple task for the study of schizophrenia is that the attention deficit can be observed within a subject. The subject's performance in the left visual field and right visual field can be compared within the exact same task format. This result eliminates such explanations as lack of motivation, fatigue, and other general reasons that schizophrenics might differ from normals in task performance.

What might be the anatomical and psychological explanation underlying such a right visual field deficit of attention? One possibility is that a deficit of the parietal lobe accounts for the visual spatial abnormality shown in figure 10. In this case a separate anterior deficit would be needed to account for the problems with language processing that are found in the literature. There are known pathways that connect the posterior cortex and these tend to involve the basal ganglia as well (Alexander, Delong and Strick, 1986). Another possibility is a deficit involving the anterior attention system common to spatial and language processing. One reason that a deficit in the common anterior attention systems seem likely is that schizophrenics who report auditory hallucinations appear to be somewhat more likely to shown stronger visual spatial deficits. Moreover, Bick & Kinsbourne (1987) have shown that auditory hallucinations seem to be related to self generated voices by the patients. Our work with normals has suggested that it is possible to create a right visual field deficit somewhat similar to that found in schizophrenics by having them shadow auditory messages while responding to visual spatial cues.

It has been known for some time that schizophrenics have difficulty in selecting and holding a set (Weinberger, 1986). Weinberger (1986) has shown severe deficits in the Wisconsin Card Sorting Task. His work with PET shows that this task seems to be related to an area of the frontal lobe called the dorsolateral prefrontal cortex. This is an area of the brain that when lesioned in monkeys produces severe deficits in tasks involving conflicts between previously rewarded acts and current information (Goldman-Rakic, In Press). The mediation conflict between competing signals is of course a basic aspect of attentional control. To study this form of conflict we used the word-arrow version of the Stroop task described previously (page 28). We had previously shown that patients with right hemisphere lesions tend to respond well to the words but poorly to the arrows and the reverse for left hemisphere lesions. Unmedicated schizophrenics, like left hemisphere lesioned patients, show a very large preference for the arrow.

Both the word-arrow conflict results and Fig. 10 point to an anterior left hemisphere deficit that is attentional because of its strong interaction with cues. While the exact nature of the disorder of attention involved in schizophrenia remains a puzzling mystery, our results provide markers that seem to relate both to the laterality of the disorder and to its attentional nature. Within subjects markers for the schizophrenic syndrome provide us with new methods for investigating the nature of this disorder and perhaps, tieing it to the underlying anatomy of the attention system. The ability to specify the

mental operations should open up new ways of linking human disorder to the underlying physiology. Even the preliminary results support the general framework of this chapter and may aid in the search of theory-driven hypotheses about the nature of other putative disorders of selective attention.

VII. Summary

This chapter has attempted to lay out a very general empirical approach to the neuropsychology of normal attention and of its disorders. The approach uses both cognitive and anatomical data to develop a structural model of the neural systems involved in selecting an item for awareness. The major general conclusion is that the nervous system localizes cognitive operations in widely separated neural systems that are then orchestrated in performance.

To study disorders of attention one may seek links at the level of impairment of mental operations. For example, we find that schizophrenia impairs the ability to shift attention to an event in the right visual field and impairs the selection of a spatial cue. Or one may seek to link impairments in neural systems to individual operations, as in the assertion that right parietal lesions impair the ability to disengage attention to deal with a target located in a leftward direction. It is also possible to indicate the functional significance of an impairment, as for example when it is asserted that a parietal deficit impairs the ability to read.

Our analysis relates diverse methods such as cognitive experiments with normals, study of brain injury and mental disorders, and the use of neuroimaging techniques. Although our description of attention remains incomplete at both the computational and neural systems levels it already provides a basis for understanding some putative deficits in terms of their effects on the structures and functions of what we now regard as a cognitive system for the selection of information.

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Figure Captions

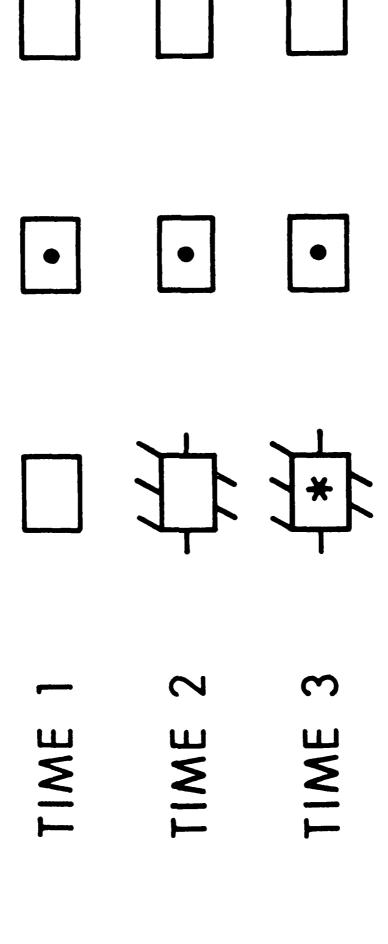
- Fig. 1. A general framework for levels of analysis in connecting cognitive tasks of daily life to neural systems.
- Fig. 2. A model task for the study of covert shifts of visual spatial attention.
- Fig. 3. The putative model operations that are set in motion by the presentation of a peripheral cue.
- Fig. 4. Three forms of neglect. The left panel shows performance when targets are in the non neglected visual areas. The right panel when they are in the neglected visual areas. Data are always from cue to target intervals of 100 msec or less.
- Fig. 5. Performance of ten right parietal patients on word and non word strings presented to them on cards shortly after their lesion.
- Fig. 6. Subtracted PET images of cerebral blood flow. The stimulated task involves visually presented words presented at fixation at the rate of one per second. The control condition is fixation alone. Both conditions involve an average over 40 seconds.
- Fig. 7. Subtracted PET images of cerebral blood flow. The stimulated task involves reading visually presented words. The control task is the passive reception condition described in Fig. 6.

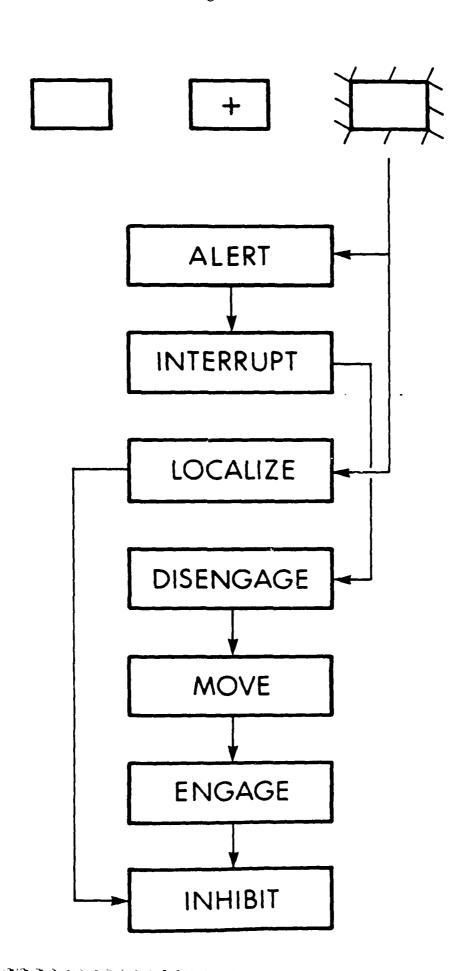
- Fig. 8. Subtracted images of images of cerebral blood flow averaged over ten subjects. The stimulated state is the generating a use for each presented noun stimulus. In the stimulated state each word is presented as described in Figure 8. (From Petersen, et al, 1987).
- Fig. 9 A hierarchically distributed view of selective attention to spatial and language stimuli.
- Fig. 10. Reaction times of never medicated schizophrenics, medicated schizophrenics and normals in the model cueing task for visual spatial attention. All data are from the 100 millisec target to cue interval.

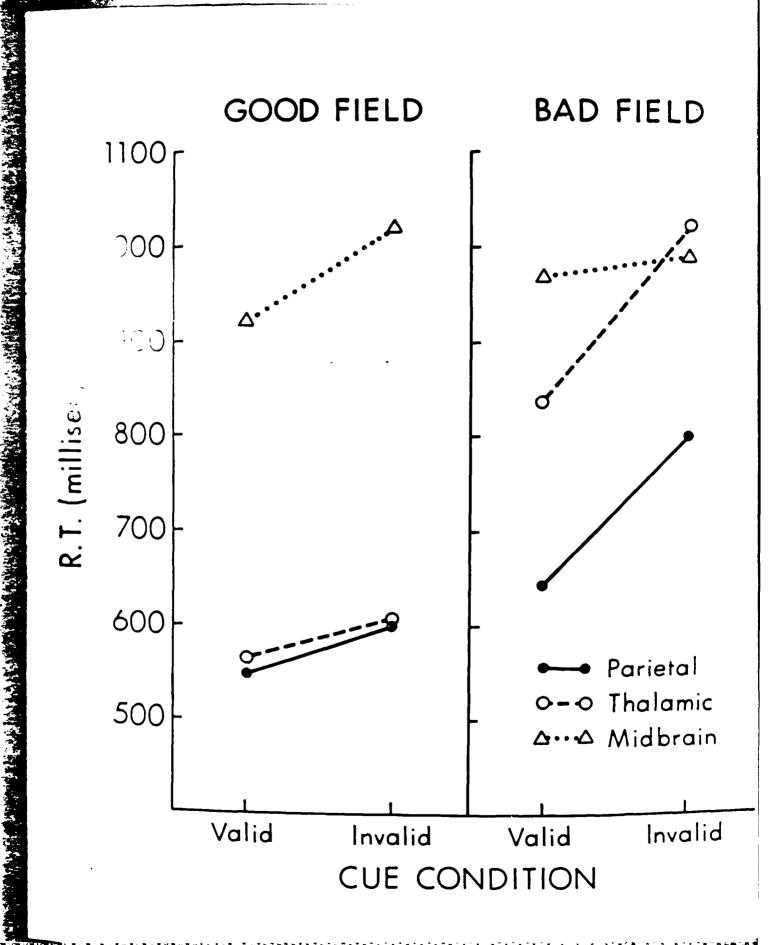
Figure 1

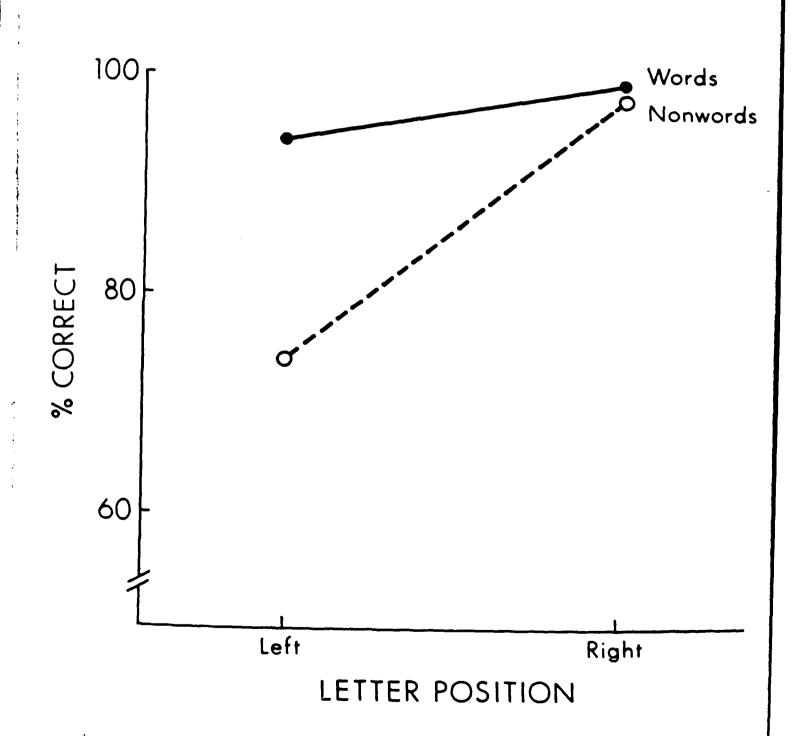
COGNITION AND NEURAL SYSTEMS

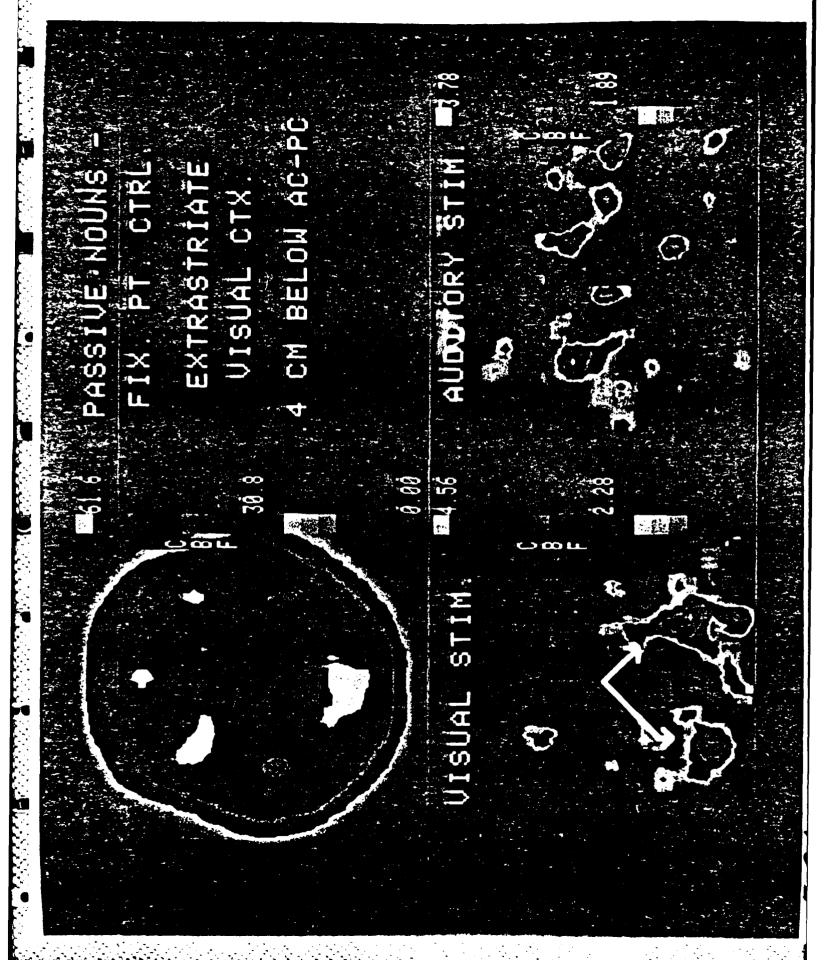
COVERT ORIENTING TASK EXAMPLES	COVERT ORIENTING	DISENGAGE, MOVE, ENGAGE	FACILITATE LOCATION	MIDBRAIN (SUPERIOR COLLICULUS) PARIETAL LOBE	LIGHT SENSITIVE CELLS
GENERAL	READING, SPEAKING IMAGERY	NEXT, SCAN, NAME, ZOOM	FACILITATE PATHWAY INHIBIT PATHWAY	PROCESSING NEGATIVITY BLOOD FLOW LESIONS	SELECTIVE ENHANCEMENT
LEVEL	TASK	ELEMENTARY OPERATIONS	COMPONENT ANALYSIS	NEURAL SYSTEM	CELLULAR ACTIVITY

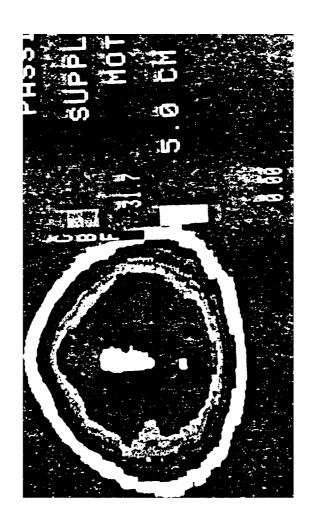


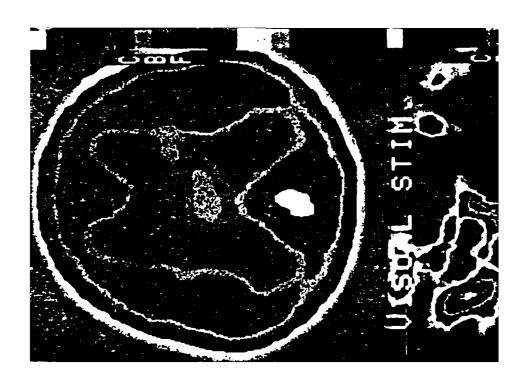




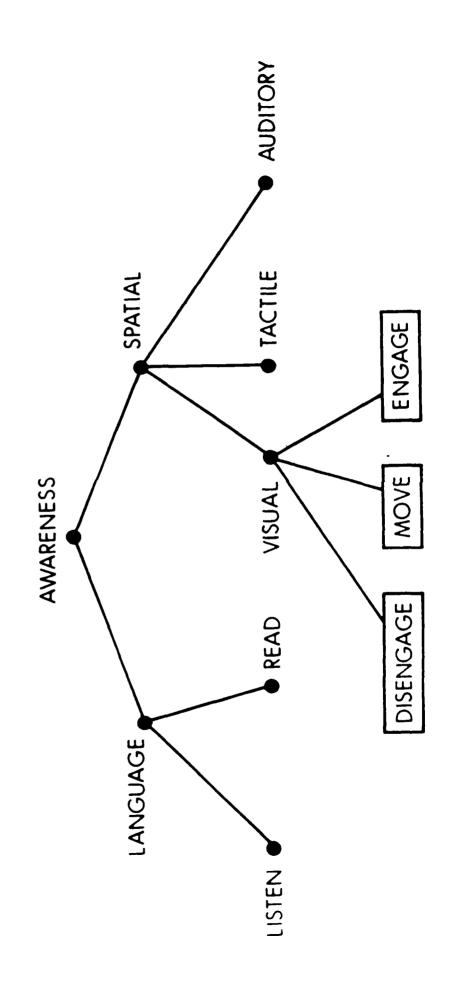


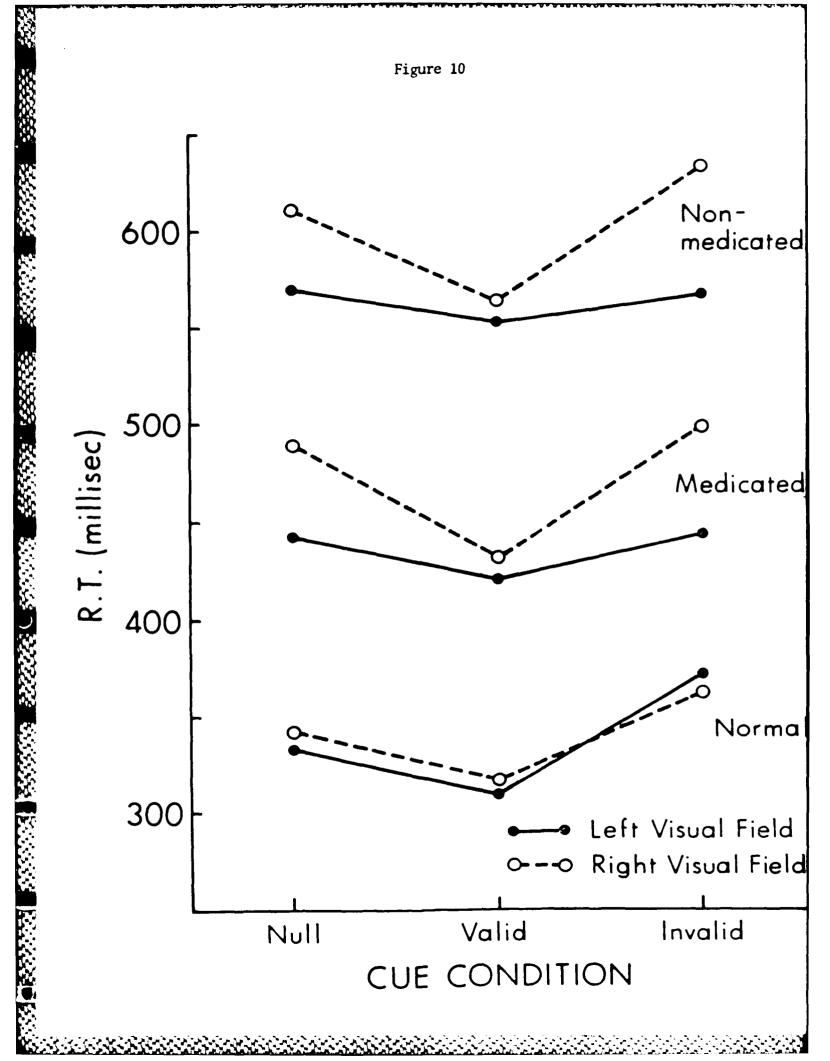






HIERARCHICAL SELECTIVE ATTENTION





References

- Alexander, G.E., Delong, M.R., & Strick, P.L. Parallel organization of functionally segregated circuits linking basal ganglia and cortex. Annual Review of Neuroscience, 1986, 9, 357-381.
- Allport, D.A. (1980) in Cognitive Psychology: New Directions (Claxton, G., ed). pp. 112-153, Routledge and Keegan Paul.
- Bashinski, H.S. & Bachrach, V.R. (1980) Enhancement of perceptual sensitivity as the result of selectively attending to spatial locations. Perception and Psychophysics 28, 241-248.
- Baynes, K., Holtzman, J.D. & Volpe, B.T. (1986) Components of visual attention: alterations in response pattern to visual stimuli following parietal lobe infarction. Brain 109, 99-114.
- Berlucchi, G. & Rizzolatti, G. eds. (1987) Selective visual attention, Neuropsychologia, 25A.
- Bick, P.A. & Kinsbourne, M. (1987) Auditory hallucinations and subvocal speed in schizophrenic patients. American Journal of Psychiatry, 144:22-225
- Carr, T.H. & Pollatsek, A. (1985) Recognizing printed words: A look at current models. In D. Besner, T.G. Weller & G.E. MacKinnon (eds.) Reading Research, New York: Academic Press 2-73.
- Cowey, A. (1985) Aspects of cortical organization related to selective attention and selective impairments of visual perception: A tutorial review. In Posner, M.I. & Marin, O.S.M. (eds.) Attention and Performance XI. Hillsdale, N.J.: Erlbaum, 14-62.
- Crick, F. (1984) Function of the thalamic reticular complex: the search light hypothesis. Proceedings of the National Academy 81, 4586-4590.
- DeRenzi, E. (1982) Disorders of Space Exploration and Cognition. John Wiley, New York.
- DeRenzi, E., Gentilini, M. & Pattacini, F. Auditory extinction following hemisphere damage. Neuropsychologia, 1984, 22, 733-744.
- Deuel, R.M. & Collins, R.C. The functional anatomy of frontal lobe neglect in the monkey: behavioral and quantitative 2 DG studies. Annals of Neurology, 1984, 15, 521-529.
- Diamond, A. (1987) Development of progressive inhibitory control of action: retrieval of a contiguous object. Paper given to Society for Research in Child Development, Baltimore, MD, April 1987.
- Downing, C.J. & Pinker, S. (1985) in Attention and Performance XI (ed.) Posner, M.I. & Marín, O.S.M. Erlbaum: Hillsdale, N.J. 171-187.
- Duncan, J. (1986) Disorganization of behavior after frontal lobe damage. Cognitive Neuropsychology 3, 271-290.

- Early, T.S., Reiman, C.M., Raichle, M.E. & Spitznagel, E.L. (1987) Left globus pallidus abnormality in never-medicated patients with schizophrenia. Proceedings of the National Academy 84, 561-567.
- Friedrich, F.J., Walker, J. & Posner, M.I. (1985) Effects of parietal lesions on visual matching: implications for reading errors. Cognitive Neuropsychology, 1985, 2, 253-264.
- Geschwind, N. (1965) Disconnection syndrome in animals and man. Brain, 88:237-294.
- Goldman-Rakic, (In Press) Circuitry of primate prefrontal cortex and regulation of behavior by representational analysis. In Plum, F. & Mountcastle, V. (eds.) Higher Cortical Function Amer. Physiological Society Handbook of Physiology, 5, 373-417.
- Hughes, H.C. & Zimba, L.D. (1985) Journal of Experimental Psychology: Human Perception and Performance 11, 409-430.
- James, W. Principles of psychology (Vol. 1) New York: Holt, 1890.
- Jonides, J. (1981) in Attention and Performance (Vol. IX) (Long, J. and Baddeley, A., eds). pp. 87-207, Erlbaum.
- Jonides, J. & Mack, R. On the cost and benefit of cost and benefit. Psychology Bulletin, 1984, 96, 29-44.
- Kosslyn, S.M. (1980) Image and Mind. Harvard Press, Cambridge, MA.
- LaBerge, D.L. & Samuels, J. (1974) Toward a theory of automatic word processing in reading. Cognitive Psychology, 6, 293-323.
- LaDavas, E. (1987) Is hemispatial deficit produced by right parietal damage associated with retinal or cravitational coordinates. Brain. 110, 167-180.
- Mangun, G.R., Hansen, J.C. & Hillyard, S.A. The spatial orienting of attention: sensory facilitation or response bias? ONR Technical Report SDEPL 001, December 1986.
- Marcel, (1983) Conscious and unconscious perception. Cognitive Psychology, 15, 238-300.
- Maylor, E. A. (1985) in Attention and Performance (Vol. XI) (Posner, M.I. and Marin, O.S.M., eds), pp. 189-204, Erlbaum.
- McClelland, J.L. & Rumelhart, D.E. (1986) Parallel distributed processing, explorations in the microstructures of cognition, Volume 1: Foundations.
- Mirsky, A.F. & Duncan, C.C. Etiology and expression of schizophrenia: neurobiological and psychosocial factors. Ann. Rev. Psych. 1986, 37, 291-319.
- Mishkin, M., Ungerleider, L.G. & Macko, K.A. (1983) Object vision: Two cortical pathways. Trends in Neuroscience, 6:414-417.

- Morrow, L.A. & Ratcliff, G. (1987) Attentional mechanisms in clinical neglect. Journal of Clinical and Experimental Neuropsychology, Vol 9, Number 1, (Abstract).
- Mountcastle, V.B. (1978) Brain systems for directed attention. Journal Royal Society of Medicine, 71, 14-27.
- Nagel-Leiby, S., Buchtel, H. & Welch, K.M.A. (1987) Right frontal and parietal lobe contributions to the process of directed visual attention and orientation. Journal of Clinical and Experimental Neuropsychology, Vol. 9, Number 1, (Abstract).
- Neely, J., Keefe, D. & Ross, K. (1986) Retrospective postlexical processes produce the proportion effect in semantic priming. Paper presented at the meeting of the Psychonomics Society, New Orleans, Louisiana.
- Petersen, S.E. Robinson, D.L. & Morris, J.D. (1987) Contributions of the pulvinar to visual spatial attention. Neuropsychologia, 25, 97-105.
- Posner, M.I. (1978) Chronometric Explorations of Mind. Lawrence Erlbaum, Hillsdale, New Jersey.
- Posner, M.I. (1980) Orienting of attention. Quarterly Journal of Experimental Psychology 32, 3-25.
- Posner, M.I. (1982) Cumulative development of attentional theory. American Psychologist, 32:53-64.
- Posner, M.I. (1986) A framework for relating cognitive and neural systems. EEG and Clinical Neurophysiology, Supplement 38, 1986, 155-166.
- Posner, M.I., Choate, L.S., Rafal, R. D., & Vaughn, J. (1985) Inhibition of return: Neural mechanisms and function. Cognitive Neuropsychology 2, 211-228.
- Posner, M.I. & Cohen, Y. Components of attention. In H. Bouman and D. Bowhuis (eds.), Attention and Performance X. Hillsdale, N.J.:Lawrence Erlbaum, 1984, 55-66.
- Posner, M.I., Cohen, Y., Choate, L., Hockey, R. & Maylor, E. (1984) Sustained concentration: passive filtering or active orienting. In (Kornblum, S. and Requin, J., eds). Preparatory States and Processes, pp. 49-65, Erlbaum.
- Posner, M.I., Cohen Y. & Rafal, R.D. (1982) Philosophical Transaction Royal Society of London Series B. 2908, 187-198.
- Posner, M.I., Early, T., Crippin, P. & Reiman, E. Does Schizophrenia involve a left hemisphere deficit of attention? In preparation.

- Posner, M.I., Inhoff, A.W., Friedrich, F.J. & Cohen, A. (1987) Isolating Attentional Systems: A Cognitive-Anatomical Analysis. Psychobiology
- Posner, M.I., Walker, J.A., Friedrich, F.J. & Rafal, R.D. (1984) Effects of parietal lobe injury on covert orienting of visual attention. Journal of Neuroscience 4, 1863-1874.
- Posner, M.I. & Henik, A. (1983) Isolating representational systems. In J. Beck, B. Hope and A. Rosenfeld (Eds.), Human and Machine Vision, New York: Academic Press, 1983, 395-412.
- Posner, M.I. & Marin, O.S.M. (Eds.) (1985) Attention and Performance XI: Mechanisms of Attention. Hillsdale, N.J.: Lawrence Erlbaum
- Posner, M.I. & Presti, D. (1987) Selective attention and cognitive control. Trends in Neuroscience, 10, 12-17.
- Posner, M.I. & Snyder, C.R. (1975) Facilitation and inhibition in the processing of signals. Attention and Performance V. New York: Academic Press, 669-681.
- Prinzmetal, W., Presti, D. & Posner, M.I. (1986) Does attention affect feature integration? Journal of Experimental Psychology: Human Perception and Performance 12, 361-369.
- Raíal, R.D. & Inhoff, A.W. (1986) Midbrain mechanisms for orienting visual attention. In: Program of the Eighth Annual Conference of Cognitive Science Society, London:Lawrence Erlbaum, Abstract.
- Rafal, R.D. & Posner, M.I. (1987) Deficits in Visual Spatial Attention Following Thalamic Lesions, In preparation.
- Rizzolatti, G., Riggio, L., Dascola, I. & Umilta, C. Reorienting attention across the horizontal and vertical meridians: evidence in favor of a premotor theory of attention. Neuropsychologia, Volume 25, Number 1A.
- Roland, P.E. Cortical organization of voluntary behavior in man. Human Neurobiology (1985) 4:155-167.
- Rumelhart, J.L. & McClelland, D.E. (1981) An interactive activation model of context effects in letter perception: part 1. An account of basic findings. Psychological Review, 88, 375-407.
- Rumelhart, D. & Norman, D.A. Simulating a skilled typist. Cognitive Science, 1982, 6, 1-36.

- Sandson, J. & Posner, M.I. (1987) Effects of divided attention on identity and semantic priming. ONR Technical Report.
- Sieroff, E. & Michel, F. (1987) In right/left hemisphere patients and the problem of lexical access, Neuropsychologia.
- Sieroff, E., Pollatsek, A. & Posner, M.I. (1987) Recognition of visual letter strings following injury to the posterior visual spatial attention system, Cognitive Neuropsychology.

- Sieroff, E., & Posner, M.I. (1987) Cueing spatial attention during processing of words and letter strings in normals, Cognitive Neuropsychology.
- Taylor, D.A. (1977) Time course of context effects, Journal of Experimental Psychology, 106:404-426.
- Titchner, E.B. (1908) Lectures on the elementary psychology of feeling and attention. New York: Macmillan.
- Treisman, (1987) Features and objects in visual processing. Scientific American, 114-125.
- Walker, J.A., Friedrich, F.J., & Posner, M.I. (1983) Spatial conflict in parietal lesions. Paper presented to International Neuropsychology Society, San Diego.
- Weinberger, D.R. (1986). The pathogenesis of schizophrenia. In Nasrallah, H.A. Weinberger, D.R. (eds.) The Neurology of Schizophrenia, 397-407.
- Wurtz, R.H., Goldberg, M.E. & Robinson, D.L. (1980) Progress in Psychobiology and Physiological Psychology, 9, 43-83.

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